rial. The profession has been too slow in taking up the use of finer suture materials. As the contraction of tendons is greater than nerves, the flexion of the joints adds very materially in overcoming this temporary defect.

The treatment of injuries to the hand, particularly in the injuries to the metacarpals, is most excellently brought out. The particular stress laid on the early treatment in minor injuries to the hand is important, and the method and care of a debridement of the wounds is most clearly elucidated.

I am impressed that in an infection of the finger nail no deformity will follow or remain after the removal of the nail if the matrix has not been injured.

Great stress has been laid on the care of opening abscesses of the hand and preservation of the nerve supply. This is particularly important as the nerves are mostly sensory in type and their destruction is followed by great disability and atrophy of the hand which largely impair its function.

Attention has been called to the site of opening palmar abscesses as the lateral side of the tendon sheath. The paper clearly points that the sheath and tendon are not involved and if the operating surgeon is careful, infection will never be carried into this anatomical structure.

The use of local anesthetic at some distance from the area of infection for the purposes of nerve block is a recommendation that all may profitably follow.

The use of heat in the stimulation of tissue repair or absorption of exudate and transudate is very important, but should not be excessive and cook the tissues. Neither should antiseptics be so strong as to be escharotic. Inasmuch as the hand may be worth from $1800 to $25,000 a year to the injured patient, Doctor Bunnell wisely recommends early hospitalization in hand cases. This is worthy of more emphasis; there is so much to be gained, and everything to be lost.

SUBACUTE BACTERIAL ENDOCARDITIS*

PART I

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WE learn from the medical dictionary that the term "endocarditis" means inflammation of the endocardium, whether it be the endocardium which lines the walls of the heart chambers or that which covers the valve cusps. It is obvious that it does not include the large group of lesions which are commonly called chronic endocarditis, in which there is inefficiency of heart function because of distorted heart valves, the result of healing of an inflammatory process which has ceased to be active. It is unfortunate that the use of the term "endocarditis" in this sense has become so general because it misleads as to the actual pathology which exists in the heart, and it necessitates specific definition whenever the word is used. One does not speak of a deformed extremity, the result of healing of a fracture with the bone fragments in malposition, as a chronic fracture, nor of the deformity which is produced by contracture following healing of an extensive burn as a chronic burn. It is no more logical to speak of deformed heart valves in which there is no active inflammatory process as chronic endocarditis, and in this report such lesions will be described as chronic valvular disease or healed endocarditis.

Again, when one discusses bacterial endocarditis, one must exclude all cases in which the disease of the endocardium is not of bacterial etiology. The most obvious group of this type is that in which the infecting organism is the treponema pallidum, but there is some question as to whether rheumatic endocarditis should not also be excluded. Despite the claims of Poynton and Payne [1] as well as of Coombs [2] and other authors, it is not definitely established that the endocarditis of rheumatic fever is of bacterial etiology, but for the sake of completeness it has been included in the proposed scheme of classification because it illustrates a peculiar type of endocarditis which may be of bacterial etiology.

The terms "acute," "subacute," and "chronic" are also commonly used in ways which are confusing. They may be used to indicate elapsed time, that is, the duration of illness, but they may also indicate the intensity or type of the inflammatory reaction in the tissues. It is true that there is often a close relationship between the duration of the illness and the intensity of reaction, but this is not constant in bacterial endocarditis. Wauchope [3] in a recent report states that in her series it was impossible to draw clean-cut lines between acute, subacute, and chronic infective endocarditis, either on clinical or pathological lines, and she adopted a time differentiation in describing her cases. She considered as acute all cases in which the clinical course was less than three months, unless—and this is important—there was postmortem evidence of organization of the vegetations on the valves, scarred infarcts, etc., in which case she classified them as subacute without regard to the duration of illness. Cases of from three to six months' duration she classified as subacute, and those of more than six months' duration as chronic.

Although this question is open to difference of opinion it would seem to be preferable to classify in terms of virulence of infecting organism and degree of reaction to infection, and in this report the terms, "acute," "subacute," and "chronic" are considered from that point of view. No differences in the duration of the illness are implied.

With these points in mind the following scheme of classification is proposed. It is not perfect, but may have some merit in that it emphasizes the virulence of the infecting organism and basic pathological processes as the indices for differentiation.

CLASSIFICATION

Bacterial endocarditis is an active inflammatory process caused by infection with bacteria in which the endocardium is the seat of the disease. The heart muscle is always, and the pericardium is usually, coincidentally affected in some degree. It adds much to the clarity of description if the name of the infecting organism is added, thus, acute pneumococcus endocarditis, subacute nonhemolytic or anhemolytic streptococcus endocarcarditis, and other conditions.

A. Endocarditis caused by infection of low virulence:

The inflammatory process is productive or proliferative and not destructive, and the tendency is to local healing and clinical recovery. Rheu-

* Part II of the paper will appear in the February issue of this journal.

TABLE 1.—*Relative Duration of Illness Among Cases of Bacterial Endocarditis*

DURATION	Pneumococcus Thayer CASES	%	Staphylococcus aureus, Thayer CASES	%	Gonococcus Thayer CASES	%	Other Streptococci, Thayer CASES	%	Streptococcus viridans, Thayer CASES	%	Streptococcus viridans, Dickson and Cooke CASES	%
0-1 week	6		8		0		16		0		0	
1-2 weeks	7		6		0		7		1		0	
2-3 "	7		5		2		5		1		0	
3-4 "	3		3		7		4		2		0	
	23	82.1	22	84.6	9	39.1	32	74.8	4	8.7	0	0.0
1-2 months	3		0		6		2		4		5	
2-3 "	1		1		4		2		1		9	
3-4 "	0		0		1		1		7		6	
4-5 "	0		2		1		2		7		7	
5-6 "	1		1		0		1		6		10	
	5	17.8	4	15.3	12	52.1	8	18.8	25	54.3	37	56.9
6-7 "	0		0		0		1		5		2	
7-8 "	0		0		1		0		1		1	
8-9 "	0		0		1		1		2		3	
9-10 "	0		0		0		1		4		3	
10-11 "	0		0		0		0		0		1	
11-12 "	0		0		0		0		2		2	
	0	0.0	0	0.0	2	8.7	3	6.4	14	30.4	12	18.5
Over 1 year	0		0		0		0		3	6.5	9	13.8
Unknown											7	10.8
TOTAL	28		26		23		43		46		65	

matic fever endocarditis is of this type, and it is unusual if a patient dies during the active stage of the disease unless there is mixed infection. In the process of local healing, however, there may be distortion and shrinkage of the valves resulting in permanent incompetency, even when actual healing is complete. It is because of the extra strain upon the myocardium due to the incompetent valves, and not because of progressive inflammatory processes in the endocardium, that cardiac failure may eventually develop.

B. Endocarditis caused by infection of moderate virulence:

There is considerable variation in the degree of virulence and in the duration of illness in this group, some cases being of a septic type with widely swinging temperature and frequent chills, while others run an uneventful course with low fever, no chills, and no signs of virulent infection. From the standpoint of pathology the process is progressive and is more or less destructive, but there is no rapid suppuration and there is a tendency to local healing behind the active process. Some authors, notably Libman,[4a] have described clinical recovery in a few instances, but all agree that the majority of patients die after a progressive course of from three months to a year after clinical diagnosis is possible.

Streptococcus viridans and *Bacillus influenza* † are the etiological factors in the vast majority of cases of this group, but a few cases of infection with gonococcus and, more rarely, with other types of streptococcus and pneumococcus may closely simulate this type in clinical course and pathological process.

C. Endocarditis caused by infection of high virulence:

The pyogenic bacteria, pneumococcus, staphylococcus, gonococcus and streptococcus pyogenes, are responsible for cases of this type, and in the great majority, with the possible exception of gonococcus endocarditis, the process is truly malignant and of relatively short duration. In most of these cases the endocarditis is not primary but is secondary to or metastatic from primary infection elsewhere, puerperal sepsis, pneumonia, osteomyelitis, etc. The process is typically destructive, there is no tendency to heal in the majority of cases and the progress of the disease is rapidly fatal, few patients surviving for more than a few weeks.

Table 1 shows the relative duration of illness in endocarditis of Groups B and C. Those produced by pneumococcus, staphylococcus aureus and "other" types of streptococcus are rapidly fatal; those caused by gonococcus occupy an intermediate position, and those caused by streptococcus viridans are the least rapidly fatal.

The material upon which this study is based consists of sixty-four fatal cases of subacute streptococcus endocarditis; twenty-six in which diagnosis was made from the clinical course of the disease and positive blood culture, and thirty-eight which came to necropsy. The series does not include a number of cases in which clinical diag-

†At least two cases have been reported in which Brucella abortus was isolated from the lesions at necropsy. These raise the question as to whether those described as being due to infection with Bacillus influenza may not have been caused by Brucella abortus.

nosis was not confirmed nor seventeen necropsy cases which fall under Group C of our classification. All of the cases occurred in the Stanford medical services at Lane Hospital and the San Francisco Hospital, or were private patients who were seen in consultation by the author or for whom the bacteriological examinations were made by him. With two or three exceptions, all blood cultures were taken by him or by his assistants, and all necropsies were performed by Doctor Ophüls or his associates.

All of the positive blood cultures showed growth of anhemolytic streptococci, seventeen of which were classified at the time as viridans. It is probable that some of the others may have shown green pigment, but they were studied before the viridans nomenclature was introduced into the laboratory.

INCIDENCE

The incidence of this disease is probably greater than is usually believed. Libman [4b] states that 0.3 per cent of persons with chronic valvular disease die of subacute bacterial endocarditis, and Horder [5] found that one in every one hundred and seventy-three patients in his ward service suffered from this disease. There is some question as to whether the disease is becoming more common, but the question cannot be answered from this small series. Cotton [6] recorded that 8 per cent of invalided soldiers with gross valvular lesions had subacute bacterial endocarditis, and Morawitz [7] stated that it had been observed more frequently in Germany since the war. The most recent data on this subject are those of Wauchope,[3] who found that in London hospitals the disease became much more common immediately after the war although the increase was due to the incidence in ex-soldiers, whereas that among civilian males and females remained unchanged.

Forty-eight males and sixteen females, respectively 75 and 25 per cent, made up our series. This corresponds fairly closely to Blumer's series [8] of three hundred and twenty-eight collected cases in which 60 per cent were males and 40 per cent were females. There is nothing in our records to suggest racial susceptibility, but in Thayer's series at Johns Hopkins Hospital [9] there is evidence that negroes are especially susceptible to gonococcus endocarditis which may simulate the viridans type in some degree.

Forty-four of our patients, 71 per cent of those in whom the age is recorded, were between twenty and forty-nine years of age, five were between ten and nineteen, and six were over sixty. Taking for comparison the incidence between ten and forty-nine, our series shows 79.3 per cent as compared with 86 per cent in Blumer's series.[8]

Among the predisposing causes, chronic valvular disease of the heart appears to play an important part although the reason for this is not clear. It is suggested that in healed endocarditis the vascular arrangement is such as will favor the lodging of small emboli and thus determine the focus of new infection. Eighteen patients in our series gave a history of one or more attacks of rheumatic fever, and one of chorea. Seventeen gave history which indicated old, mostly long-standing, heart lesions and one, the youngest in the group, had a congenital heart lesion.

ONSET

The majority of records contain no reliable data as to the immediate source of infection, but in our series the beginning of the terminal illness occurred in three patients immediately after the extraction of abscessed teeth, in one during convalescence from an acute infection which was diagnosed pneumonia, in one after curettage in a woman who had induced an abortion two years before and had had persistent vaginal discharge since then, and in one following an infected finger. At least ten patients stated that they had caught a severe cold or had "influenza" several weeks or months before and had not been able to regain their strength. One very interesting case in this last group was a young woman who for three years had shown typical signs and symptoms of Addison's disease, which, incidentally, was proved at necropsy to be associated with extensive tuberculous lesions in the adrenals. About eight months before admission to hospital she had an attack of "influenza" after which she was unable to work, and lost ground rapidly. One patient who was pregnant had recurring attacks of bronchitis, tonsillitis and arthritis during pregnancy and died before reaching term.

A number of cases were found at necropsy when clinical diagnosis had not been made. The most interesting of these was the case of a woman who had had diabetes for several years and who entered the hospital in coma. She responded fairly well to insulin but collapsed suddenly and died when insulin was discontinued.

The lesions of the patient who had Addison's disease conformed to those of Libman's [4a] terminal cases in that there were no signs of embolism and no bacteria were found in smears from the lesions on the heart valves, but in the other case, the patient with diabetes, smears from the heart-valve lesions contained streptococci and there were many recent infarctions in the kidneys and spleen.

PATHOLOGY

The pathology of subacute bacterial endocarditis must be considered from three points of view:

1. The changes in the heart.
2. The changes due to long-standing infection.
3. The changes due to embolism.

The heart may be of normal size, or may be large, particularly in those cases in which there has been aortic insufficiency of long standing. There may or may not be hydropericardium, but in our series the amount of fluid was never very great. Usually the pericardium is smooth, and often studded with petechiae, but in five cases there was fibrinous pericarditis, four general and one localized, all of them associated with aortic valve lesions. No perforations through the heart wall were demonstrated, but it seems quite probable that the pericardial process was secondary by extension from the lesions at the base of the heart.

The heart muscle is usually opaque and may have necrotic areas if there has been embolism

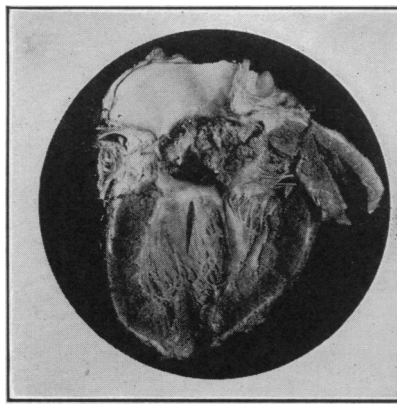

Fig. 1 (Case 29—57).—Note the deep lesions at the aortic orifice and the fibrinous pericarditis.

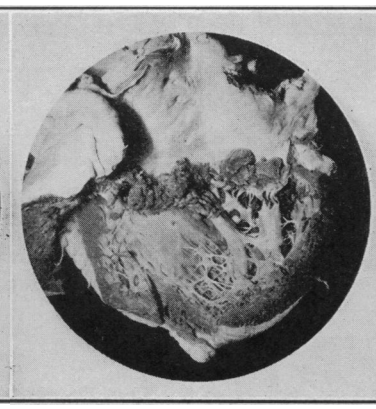

Fig. 2 (Case 18—166).—Note the involvement of the mitral valve with extension to the chordae tendineae.

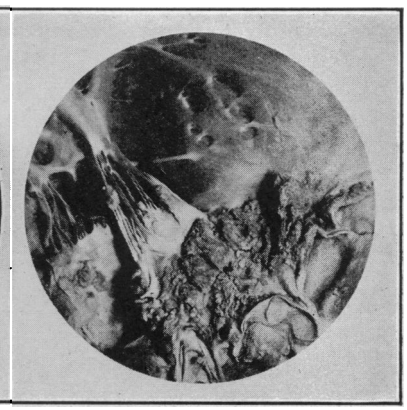

Fig. 3 (Case 16—205).—Note the lesions on the proximal and distal mitral cusps, the chordae tendineae, the aortic cusps, and the wall of the ventricle.

into the coronary arteries, but ordinarily it is fairly firm and does not show extensive signs of tissue degeneration.

The endocardium, however, shows very extensive and very characteristic lesions. They are involvement of the valves, of the chordae tendineae and sometimes of the tips of the papillary muscles, and of the mural endocardium. The lesions in the valves tend to be proliferative rather than destructive and the typical vegetation is a firm, fibrinous mass, sometimes small and sessile, at other times large and polypoid or even massive. There may be extensive destruction of the flaps of the valves and there may be aneurysms or perforation of the valves; in fact, the common coincidence of aortic and mitral valve lesions is often due to perforation of the large flap of the mitral valve and extension of the process on the surface opposite to that bearing the initial lesion. The vegetations may be pale or pink in color and there is always an attempt at healing at the base. The extent of healing varies greatly, and Libman[4c] has reported cases in which it has been complete. In our series there have been the usual variations in degree, often with calcification at the bases of the vegetations, but in one case there was evidence of an old extensive process of the mitral valve, involving the chordae tendineae and the tips of the papillary muscles, which was entirely healed; the terminal acute illness was associated with a large, recent mural involvement in the right auricle.

Libman[4a] emphasizes the fact that the vegetations of the subacute type are usually larger than in rheumatic fever, and that there is absence of the typical Aschoff bodies in the myocardium. There are, however, so-called Bracht-Waechter bodies in subacute cases, round cell interstitial lesions which occur in the myocardium. There is usually no evidence of pus formation in lesions caused by emboli in the coronary arteries. The characteristic incidence of the disease in adult life would indicate that in the majority of cases the lesions are of the valves of the left side of the heart, and this is in fact the case. A survey of our series shows approximately the same distribution as was found by Blumer[8] in his survey of the literature, and is in sharp variance with the distribution in rheumatic endocarditis where there is a high incidence of tricuspid involvement.

Extension to the chordae tendineae occurred in nine of the twenty-nine cases in which the mitral valves were involved, and there was involvement of the mural endocardium in nine. In one instance the only involvement of the right side of the heart was an extensive mural vegetation which was secondary to, and directly continuous with a lesion in the left ventricle which had perforated the interventricular septum.

The manifestations of general infection are varied, the most important being enlargement of the spleen, secondary anemia and cloudy swelling of the parenchymatous organs. There is a tendency to diffuse hemorrhages in the serous surfaces and in the skin in addition to the presence of petechiae which are embolic, and one of our patients showed pachymeningitis interna hemor-

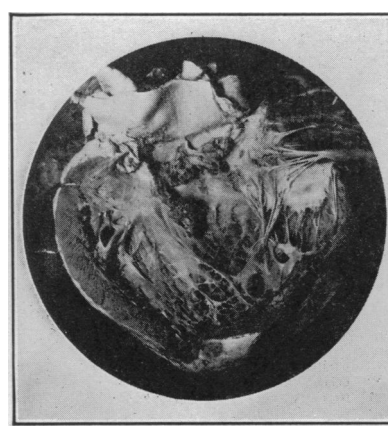

Fig. 4 (Case 28—55).—Note the lesions of the aortic cusps and the extension to the wall of the ventricle.

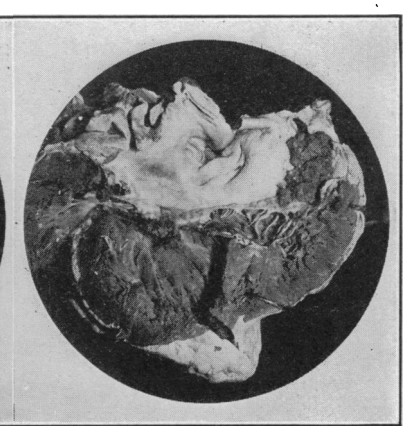

Fig. 4 (Specimen 52—24).—Note the healed lesion on the mitral valve and the active process on the wall of the auricle.

TABLE 2.—*Distribution of the Active Processes in the Heart in Subacute Bacterial Endocarditis*

Valves Affected	Dickson and Cooke 38 Necropsies		Blumer 150 Necropsies*	
Mitral valve only	13	92.1%	58	89.0%
Aortic valve only	6		16	
Mitral and aortic valves	16		56	
Pulmonary valve only	–		4	
Tricuspid valve only	1		3	
Mitral and tricuspid valves	–		1	
Aortic and tricuspid valves	–		3	
Aortic and pulmonic valves	–		1	
Mitral, aortic and tricuspid valves	1		4	
Chordae tendineae affected	9			
Walls of chambers affected:				
Left auricle	4			
Right auricle	3			
Left ventricle	4			
Right ventricle	2			

* Collected from the literature.

rhagica for which no local cause could be determined. The spleen was enlarged in twenty-five of our necropsy series.

As already stated, there are often showers of petechiae in the skin and conjunctival membrane which are caused by minute emboli, and similar minute emboli may lodge in the glomerular tufts in the kidneys to cause the characteristic glomerular nephritis of Löhlein[10] and Baehr.[11] Among thirty-nine clinical records which were available, nineteen recorded petechiae and in our necropsy series glomerular nephritis was recorded nineteen times. The distribution of the glomerular lesions is scattered; Gaskell[12] has described the appearance of the kidney as "flea-bitten"; and the total kidney function may not be much impaired. However, there is usually also a parenchymatous degeneration of the kidney epithelium, and if one carefully examines the urine one will find albumin, casts and red blood cells in most cases.

Embolism due to larger fragments than produce petechiae and glomerular lesions are very common, and occurred in twenty-seven cases of our necropsy series. The greatest number lodged in the vessels of the spleen and kidneys, respectively fifteen and sixteen times, but seven lodged in the vessels of the lungs, five in the coronary arteries, four in the vessels of the brain, three in the leg, two in the mesenteric artery, and one each in the iliac artery and an artery of the tip of the nose. In two instances embolism was the first clinical indication of serious illness, hemiplegia being caused in one and obstruction of the femoral artery in the other. In only two instances were the metastatic lesions of a purulent nature.

SIGNS AND SYMPTOMS

Blumer[8] has briefly stated the general characteristics of the disease as follows: "The clinical manifestations of subacute bacterial endocarditis naturally fall into two periods, an early period when the symptoms and signs are those of low-grade infection and a late period when the manifestations of embolism hold the prominent place. The duration of the first period is usually impossible to determine because the onset of the disease is so insidious that the exact time at which infection occurred cannot be fixed. The onset of the stage of embolism is more readily appreciated as the emboli, even when they involve the internal organs, give rise to recognizable clinical phenomena."

A typical onset of the insidious type is illustrated by the history of a butcher, age thirty-five, who believed he was perfectly well until four months before admission to hospital, when he began to be short of breath on exertion and tired easily. Within two or three weeks he was forced to cease work and soon was so weak that he remained in bed. He had no pain, did not know he had fever and was very comfortable so long as he remained in bed. He died about four months after first noticing weakness.

Eighteen of our patients complained of shortness of breath, ten of weakness, five of chills and fever, two of night sweats, four of gastrointestinal disturbances, including loss of appetite, dyspepsia and diarrhea, two of precordial pain, four of palpitation, four of swelling of the feet, and one of arthritis. One patient had passed through normal pregnancy and was apparently convalescing satisfactorily when she suddenly developed hemiplegia three weeks after delivery. Subsequent necropsy showed that this was due to embolism.

There is no regular sequence in the development of signs and symptoms, in fact, the course as well as the mode of onset varies so greatly that it is difficult to describe a typical case. The signs and symptoms may be very obscure at first, low-grade fever, pulse somewhat increased in rate, little if any impairment of nutrition, normal blood or slight anemia, weakness and early fatigue. If the physician has known the patient to have had valvular disease of the heart, or if there is history of valvular disease, his attention is not particularly aroused by the signs of valvular insufficiency unless the murmurs differ from those which he had previously heard. Suspicion is aroused only when the fever persists, when there are recurring febrile periods with short intermissions, when there is progressive anemia, the spleen becomes palpable or petechiae appear.

In addition to the early symptoms which have been mentioned there may be chilly sensations, painful erythematous nodules in the skin, pains in the muscles or joints without local reaction, backache and smoky urine. One of our patients was not alarmed about his condition until there was sufficient blood in the urine to be macroscopically visible.

A number of patients, however, have a stormy onset, with chills and fever, extreme prostration and headache. Several cases have been diagnosed as malaria, and in one instance, a professional man who had suffered from malaria, it was at least two months before he would admit that his symptoms were different from those he had experienced during his attacks of malaria.

Symptoms referable to the nervous system are common. In the early stages there is often restlessness and apprehension which, in the absence of positive objective signs, may lead to a diagnosis of hysteria. Headache is very common, sometimes occurring early, and sometimes very

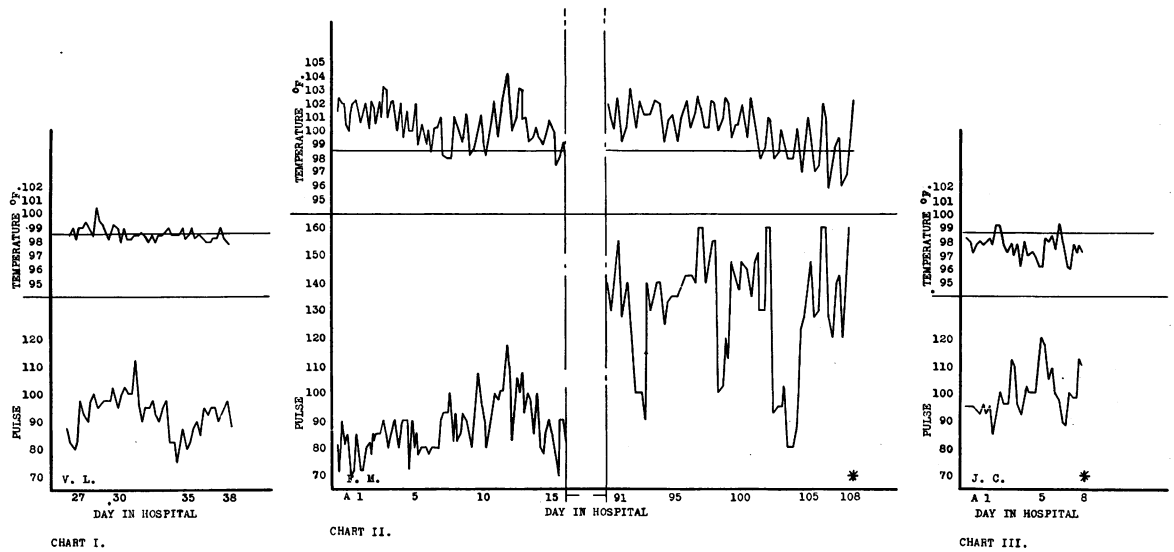

CHART I. CHART II. CHART III.

severe. Vertigo was an outstanding early feature in two of our cases. Many of the patients sleep poorly and become depressed, but it is surprising how many of the victims remain cheerful and confident of recovery, their only distress being that they are so weak they cannot be up and moving about. Rarely there is delirium in the terminal stages and there may be coma before death. Convulsive seizures, coma and hemiplegia may be caused by emboli and sometimes there is aphasia from embolism without signs of paralysis.

Acute meningitis is rarely seen as a result of cerebral embolism, a very striking difference from what occurs when the infectious agent belongs to the third group of our classification.

Nutrition suffers little at first and sometimes remains good throughout the disease; usually, however, there is progressive loss of weight but not to the stage of emaciation. The appearance of the skin is fairly characteristic, especially in the later stages. There is a peculiar brownish yellow discoloration, which is not jaundice, but which Libman [4d] has described as a café-au-lait tint. It differs from the lemon-yellow color of pernicious anemia, but is difficult to describe. The sclerae become progressively more pearly-white as the anemia progresses, and when petechiae appear they are especially to be found in the conjunctiva, the pharyngeal mucous membrane and the skin of the axilla, arms and chest, although they may be widely distributed. Occasionally there is true jaundice.

Examination of the chest gives little help at first unless there is a change in the character of heart murmurs which have been known to exist. Libman [4d] has described tenderness on pressure over the upper part of the sternum, but this was not at all constant in a number of our cases. There is usually some bronchitis and patients often complain of cough, but when signs of pulmonary involvement are present, they are apt to be confusing rather than helpful in diagnosis.

There is nothing characteristic in the abdomen until the spleen becomes palpable, which may be early or comparatively late in the disease. Sometimes it is enlarged when the patient first comes under observation, but at other times it is not felt for several weeks. In one patient who first became ill in November, and who was continuously under observation from early in January, the spleen was not felt until the middle of March, although his temperature was high during the whole time. The liver is usually enlarged but is not tender. In the rare instances when the condition develops upon a heart which is already verging on decompensation there may be early signs of liver engorgement.

Examination of the extremities gives little that is diagnostic unless petechial hemorrhages are present. There may be tenderness and swelling in the joints, occurring early or later, but this has been an outstanding feature in only two of our cases. The arthritis may persist or may be transient, in one of our cases it was recurrent. Blumer [8] states that sometimes streptococcus viridans may be recovered from the synovial fluid. The painful erythematous nodes of Osler [13] may be found along the shins or other long bones, but have not been at all frequent in our series.

There is no record of eye changes in our series, but in very few cases were they specifically searched for. Blumer [8] states that retinal changes are recorded in about 10 per cent of cases in which routine examination was made, and Falconer [14] found five in a series of fifteen cases in which he made special examination. Embolism may occur and there may be retinal hemorrhages. Libman [4a] states that optic neuritis may occur, sometimes resulting in optic atrophy, and he believes that it is much more frequent than has been realized.

There is no characteristic febrile reaction, the majority of cases running a low fever at first, although some have a widely swinging, septic type of fever from the beginning. Lenharz [15] believed that when the patients have septic temperature and chills the infecting organism is not streptococcus viridans, but that has not been our experience. Blumer [8] found that in the majority of cases the temperature swing does not exceed four degrees, but sometimes it may be greater. His survey also shows that among 249 patients whose records contain details, 116 had fever only, 70

fever and chills, 16 fever and sweats, and 34 fever, chills and sweats.

Some patients are afebrile, at least during the time they are under observation, and Libman [4a] points out that they may be in a bacteria-free stage and that there may be recurrence of the fever. In one of our patients there was an initial febrile period of three weeks, followed by a period of a month during which there was normal temperature, only to be followed by continuous fever for more than three months which lasted until his death. It may be said in passing that despite repeated examinations, blood culture was always negative in this case until six weeks after the onset of the second febrile period.

Still other patients, however, remain afebrile until death, as is shown in Chart 3. Unfortunately there is no record of blood culture in this case, but careful examination of smears from the valve lesions made at necropsy failed to show any bacteria.

The pulse rate in subacute bacterial endocarditis is more rapid than normal, and in patients with low temperature tends to be greater than the amount of temperature would explain. In fact, in one instance, had we paid sufficient attention to the discrepancy between temperature and pulse, it is doubtful whether we would have so readily accepted a clinical diagnosis of cerebrospinal syphilis. A few patients show irregularities or develop them during the course of the disease, but this is not the rule. The majority of patients continue to have regular pulse although the rate tends to become more rapid as the disease progresses.

A characteristic feature of the disease is progressive anemia of the secondary type which is rarely extreme. A survey of the red blood count and hemoglobin estimation of twenty-three patients taken during the last month of life is shown in Table 3. The figures compare fairly well with those of Blumer,[8] who found approximately one-third of recorded counts between three and four million with the remainder about equally divided above and below these numbers. It is surprising, however, that, despite the infection, the red blood count sometimes remains very high, above five million, and in one of our cases the cell count increased from 4,072,000 to 6,408,000 during the last two months of life, the hemoglobin increasing from 72 to 85 per cent during the same time.

There is very wide variation in the leukocyte counts. Not infrequently one of the confusing features is a leukopenia, sometimes as low as 3000 cells, which, taken with low-grade fever, strongly suggests typhoid fever. At other times the leukocyte count is very high; in our series the maximum observed was 60,000. The majority of cases, however, show leukocyte counts of from eight to fifteen thousand, and, as a rule, the polymorphonuclear increase is not relatively very great. The results of one hundred and seven counts in fifty patients is shown in Table 3.

TABLE 3.—*Summary of Blood Counts in Subacute Bacterial Endocarditis*

Red Blood Corpuscle Count on 23 Patients during Last Month of Illness		Hemoglobin		107 Leukocyte Counts on 50 Patients	
Over 6 million	1	80-90 %	2	50,000–60,000	2
5 to 6 million	–	70-80 %	5	40,000–50,000	2
4 to 5 million	6	60-70 %	3	30,000–40,000	3
3 to 4 million	10	50-60 %	4	20,000–30,000	8
2 to 3 million	5	40-50 %	3	15,000–20,000	16
1 to 2 million	–	30-40 %	4	10,000–15,000	27
Under 1 million	1	20-30 %	–	6,000–10,000	37
		10-20 %	1	5,000– 6,000	3
				4,000– 5,000	6
				3,000– 4,000	3
6,408,000		Highest Count		60,000	
900,000		Lowest Count		3,300	

The urine may be normal at first, but soon there is albumin and the sediment contains casts and red blood cells. The amount of blood is rarely sufficient to cause smokiness or a red discoloration of the urine, but it is almost always found at some time during the course of the disease. In fact, the appearance of red blood cells in the urine in febrile cases of uncertain etiology is one of the indications which should lead one to think of subacute bacterial endocarditis.

Sometimes bacteria may be isolated from the urine, and this may also aid in diagnosis. In an earlier report [16] one case was mentioned in which positive blood culture was not obtained, but streptococci of the same type as were subsequently found at necropsy were isolated from the urine during life.

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(To be continued)

FIBROSIS OF THE MYOCARDIUM*

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CHRONIC myocarditis was one of the diagnoses frequently made in the Cottage Hospital until recent years. This is true of most hospitals, and Cabot states that in the files of the Massachusetts General Hospital it almost leads in frequency.[1] The reason for this lies in the fact that clinical observations have not been carefully correlated with postmortem investigation into the state of the cardiac muscle. Any decompensation or arrhythmia has been considered by the clinician as an adequate basis for this diagnosis. Fahr in 1923 reported the findings in examination of hypertension hearts, as hypertrophy and dilatation of the left ventricle, diffuse connective tissue proliferation between the bundles as well as into them, and much less frequently cicatricial patches.[2] These changes occurred in the chambers called upon to do the increased work and apparently were not due to inflammatory processes. The condition of the coronary arteries was not mentioned. He believed no good grounds existed for assuming that chronic infections are the most productive factor in chronic myocarditis, as no one has ever experimentally produced these changes by inoculation. Following the suggestion of Bell he found that three-fourths of so-called chronic myo-

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